THE NEUROTIC DISTURBANCES AFTER JOINT AFFECTIONS.*

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IN the same manner as in late years the subject of joint affections occurring during the course of, and directly dependant upon certain diseases of the nervous system, was met with considerable attention on the part of neurologists as well as of surgeons, so also has the circulus vitiosus been completed and an amount of attention paid to nervous changes occurring consecutively to various joint affections, which upon first thought seems to be entirely disproportionate to the importance of the subject under consideration. It is to this latter class of cases that I desire to attract your attention this evening, and if I succeed in substantiating their gravity if left to themselves, and the great benefit which may be derived from a judicious course of treatment, we will have devoted our time to some purpose. When we consider how frequently joint lesions, both traumatic and idiopathic are encountered, and what proportion of them are followed by some disorder of the nervous elements surrounding the joint, we are enabled to realize the importance of this subject. It is, however, a noticeable fact that, notwithstanding the frequent occurrence of these troubles, it is only of late years that the subject has received any particular notice or attention. This apparent neglect was probably due to the reason that the casual relationship between joint affections and consecutive atrophic paralysis was not properly understood. Many subjects whose history we trace in medicine or surgery may be found alluded to in some shape or other by the old masters, so also has the prognosis of the class of cases of which we are now writing, received mention by Hippocrates,[1] but from that period to the time of John Hunter no reference of any value can be found. Hunter, however, in his complete works devotes an entire chapter to the subject of paralysis consecutive to arthritis, at the same time it is necessary to note that his description of the affection and its course is incomplete and in many points inexact. He explains the causal relationship satisfactorily to himself upon the score of sympathy. That this explanation is unsatisfactory to us, need not be stated, but whether later authors are more satisfactory, when they call these affections reflex, than Hunter was when he described them as sympathetic, is questionable. From Hunter to Malgaigne,[2] 1836, we have another lapse of year which passed dormant on the subject. From Malgaigne until the last decennium the whole literature, which is scanty, is almost exclusively French. During the last ten years those who have done most towards the enlightenment of our views upon the subject have been Weir Mitchell[3] by his studies upon the injuries to nerves, Duchenne (de Boulogne),[4] Vernenil, Sir James Parget, and finally those investigations of Charcot and his pupils, which bear directly upon the question. At present there cannot be any doubt that this paralysis which affects certain muscles or groups of muscles predominantly, and is a complication or a sequellor, of the various spontaneous or traumatic lesions of the corresponding joint, is directly due to and dependant upon these affections. This has been conclusively demonstrated not only by clinical experience but also by the experiments of Valtat[1] in 1878, and later by those of Christin.[2] Valtat has shown experimentally upon guinea pigs and dogs, that as a result of an injury to the articular, or even to the periarticular tissues, produced by irritant injections, the muscles of the entire limb, but more particularly the extensors of the joint became affected, and that the atrophy which was simple in character, came on very quickly after the injury, being equal to twenty per cent. at the end of one week, and to forty-four per cent. at the end of two weeks.

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[1] Hippocrates: "Traduction de Littre," vi., p. 69.

[2] Malgaigne: "Mémoire à l' academie royale," 1836.

[3] Weir Mitchell: "Injuries of nerves and their consequences." Phila., 1872.

[4] Duchenne: "De l' Electriscuion Localisée." Paris, 1861.

These two theses of Valtat and Christin have not met with the recognition outside of France that they deserve, and the dissertation of Valtat, which is now five years old, is certainly very little known. For this reason I will cite the following experiment in detail. It is taken from Christin's dissertation, and is exactly like the experiments performed by Valtat:

EXPERIMENT.—April 18, 1880.—A dog of the age of one month. Injection, by means of a hypodermic syringe, of three drops of oil of mustard into the right knee-joint. Rapid enlargement; severe pains upon pressure. The animal immediately flexes its leg under the abdomen. 19th. The swelling of the knee-joint has produced an œdema of the lower part of the right thigh. The animal remains almost continually in the recumbent position. 22d. The right knee remains swelled, and limping is uninterrupted. Atrophy of the affected limb becomes noticeable. 27th. The knee has decreased somewhat in size, and the limping takes place only occasionally. 31st. The animal is killed; the two limbs are severed from the body. The subcutaneous areolar tissue is almost entirely absent. The right knee is swollen, the periarticular tissues thickened and œdematous. The synovial membrane is of a deep red color; the joint surfaces themselves have retained their normal appearance ; the muscles of the right thigh are atrophied and paler than those of the healthy side. The muscles of the leg are not as much affected as those of the thigh, but they are paler and thinner than when in normal state. The various parts of the two extremities were then weighed and compared with each other, and the following results obtained :

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[1] Valtat: Thèse, Paris, 1878.

[2] Christin: Thèse, Paris, 1880.

	LEFT. WEIGHED		RIGHT. WEIGHED
Extremities	420.00	grammes.	340.
The sartorius muscle	4.00	"	2.5
The biceps, semi-tenchinosus, and semi-membraneous	65.00	"	48.00
The adductors and vastus internus	94.00	"	73.00
The triceps, græilis, and tensor fasciæ	88.00	"	63.00
Gluteal muscles	34.00	"	23.00
Gastrœnemei	25.00	"	21.5
The remaining muscles	28.00	"	24.00
The bones not scraped	83.00	"	86.00

These various muscular masses were then, after having been weighed, placed into two jars filled with ether, which was frequently changed. About a month after the death of the animal the muscles were again weighed. After remarking that the fat was almost entirely absent, it was also noted that the difference in weight between the different parts, which now consisted almost solely of muscular fibres, remained about the same.

The affections which most frequently follow affections to joints are paralysis and atrophy of the muscles, and hyperplasia of the subcutaneous connective tissue. These disorders being the most common and the most frequently encountered, have naturally met with the greatest attention. Besides these, there is another class of disorders which, although not as frequent as the paralysis and atrophy, are, nevertheless, of sufficiently frequent occurrence, either alone or as a complication, to merit being withdrawn from the neglect which has hitherto been their lot. This class embraces anæsthesia, hyperæsthesia, analgesia, hyperalgesia, also sclerosis of the skin, and neuralgias. The latter especially may become very troublesome.

The symptoms and course of the affection will be best shown by means of the cases which are detailed below, but there are a few remarks which it would be well to make now, and which may serve to elucidate the symptomatology. The nervous changes which take place after joint affections implicate three groups of nerve functions,—motility, sensation, and nutrition. The course of the affection is more or less as follows: After some lesion to a joint, slight or severe, at a variable period of time, the first thing that attracts the attention is a gradual change in the appearance of the limb. This, which prior to the injury, was firm and well rounded with the muscular eminences well marked, becomes soft and impressionable to the touch. The muscles become flattened, gradually lose their distinct outline, and finally the contour of the limb being effaced, it presents the appearance of one even mass, without any muscular eminences or depressions.

At the commencement, and sometimes even prior to the disappearance of the joint affection, the extensor muscles seem to be the ones which are most involved, but if the joint affection is of long duration, the other muscles become implicated in their turn, first and foremost, however, those muscles which surround the joint. Besides this general change which takes place in the appearance and consistency of the muscles, their contractile power is also diminished and, finally lost. This again is particularly the case with the extensors, although the other muscles are not entirely exempt. The electro-muscular excitability is occasionally unaffected, but in the great majority of cases it is decidedly lowered, both to the action of the faradic as well as to that of the galvanic current. By that I desire to express, that a much stronger current is requisite to produce contractions upon the affected limb than upon the unaffected, and indeed in some cases, where the affection has progressed very far, it is possible to obtain contractions with the strongest current only. Seeing this lowered excitability to the faradic current, we would be led to expect an increased reaction to the galvanic. This is, however, not the case. We do not find any reaction of nerve degeneration, no reversion of the normal contraction formula, but simply a lowered contractility.

Rumpf has, at the clinic of Erb, paid particular attention to the electrical reaction of the muscles in these nervous sequellæ of joint affections, involving in his cases mostly the shoulder and the knee, and his results are, as I have formulated them above, that there is a simple diminution of electrical excitability, and never any qualitative change. This, which has repeatedly been corroborated by Erb himself, constitutes a distinctive point between these muscular changes and any degenerative atrophy.

The time of appearance of this paralysis has been put down differently by various writers. Valtat, in a case of traumatic hydrarthrosis, which he reports in his thesis, has been able to detect it as early as twenty-four hours after the accident. I myself have never been able to notice any paralysis prior to the third day. One thing, however, is certain, and that is that in many cases it appears much earlier than we have been in the habit of supposing. The neurologist rarely sees these cases during the progress of the arthritis ; they are brought to him only after the paralysis is fully developed, and thus he is unable to decide at what period it actually supervened.

I expected to find elucidation upon this point in surgical reports, but have been disappointed. On the other hand, there is another class of cases where this disorder occurs very tardily. As for instance in the case which I mention below, in which the arthritis existed four months prior to the appearance of any nervous symptoms. As a rule the paralysis is followed by atrophy of the affected muscles. I say as a rule, for some writers—Descosse, Christin, and others—claim to have seen cases of paralysis without atrophy. The cases which have come under .my observation in which there did not appear to be any atrophy, were cases in which the intermuscular connective tissue and the subcutaneous areolar tissue were evidently hypertrophied, and thus obscured the atrophy which to my mind was present. This hypertrophy of the subcutaneous areolar tissue occurs in a great number of cases. The mere knowledge of its occurrence is sufficient to prevent its being mistaken for œdema.

It has impressed itself upon my mind that this hypertrophy of tissue stands in a direct ratio to the atrophy, particularly in those cases of periarthritis of the shoulder-joint, and that the greater the atrophy of the muscular substance the greater was the hypertrophy of the subcutaneous tissue. On this account many cases in which there was decided atrophy did, upon inspection, not appear to be atrophied at all. This atrophy is ascending and progressive in its course, and if not treated and its progress arrested, may, as in a case reported by Dr Révellont,[1] involve the entire half of the body. The atrophy follows the paralysis very rapidly, never, however, does it precede it. The atrophy can generally be made out by means of comparison by the eye or by the centimètre measure, which will be found sufficiently accurate for all practical purposes. Contracture of the muscles is also spoken of as forming a prominent complication; following the paralysis and atrophy at a short interval of time. Of the thirty cases which have come under my care since the first of January, 1883, and of which I have notes, none were associated with contracture. Of cases seen previous to that date I have no notes, but cannot recall its presence. At any rate I can safely say that, never having seen it myself, it is not of frequent occurrence. The disorders of sensation which are met with, either in conjunction with the disorders of motion, or alone, are hyperæsthesia and hyperalgesia, anæsthesia and analgesia ; also neuralgias. I find that these disorders of sensation, especially the hyperæsthesia and hyperalgesia, are early symptoms, sometimes lasting only for a very short period of time. Anæsthesia and analgesia, on the other hand, may be met with at any stage of the affection, and they frequently last for a long time and are very tenacious. They are naturally easily overlooked unless specially sought for.

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[1] Révellont: *Gazette des Hospitaux.* 3 août, 1878.

The neuralgia, which is met with in certain cases, and which is mentioned as occurring in three of Descosse's cases, in all of which it affected the crural, is peculiar in some of its characteristics. The pain is not a sharp lancinating one, but is dull and not easily excited by pressure. Still, the points of Valleix are easily made out. The patients complain of spontaneous pain along the course of the nerves, even without having had their attention directed to it.

There is hardly any necessity for dwelling upon the differential diagnosis between this affection and other atrophic paralyses. Once that the occurrence of such disorders after joint affections is impressed upon the mind the diagnosis becomes a question of observation and nothing more. There is, however, one disease which under certain circumstances may be confounded with it at the commencement, and that is progressive muscular atrophy. I mention this because the prognosis in the two diseases is so much at variance that we cannot be sufficiently guarded not to fall into so serious an error. Furthermore, I am convinced that many cases which have been reported as cured cases of progressive muscular atrophy were simply cases of atrophy after joint affections.

Atrophy, following affections of any of the joints of the hand, may in the commencement present no distinctive symptoms from those of progressive muscular atrophy. We, however, owe to Erb the discovery and description of degenerative reaction in the atrophied muscles of progressive muscular atrophy, which although very difficult to demonstrate clearly, is present in all cases, and it is this which constitutes the distinctive point for differential diagnosis. Erb[1] found this degenerative reaction to be present in all undoubted typical cases of progressive muscular atrophy, and Vierordt[2] has recently published a series of cases which corroborate this. Furthermore, in progressive muscular atrophy the atrophy always precedes the paresis. In the cases which are now being considered the paralysis, in my experience, always precedes the atrophy. Another point which may be serviceable is, that in the majority of these cases we also have disorders of sensation in conjunction with the disorders of motion. Sometimes a hyper-æsthesia, sometimes an anæsthesia; and even if at the time of examination we are not able to detect them, we will upon inquiry in all probability obtain a history of their previous existence. These symptoms may also occur in progressive muscular atrophy, but they are much less frequent, less marked, and always appear late in the disease. Even with these points to aid us, if in a case we are unable to detect any change of degeneration, have no clue as to the time of onset of the paralysis, and cannot find any distinct changes of sensation, we are obliged to leave the diagnosis *in suspensu*, and to allow Time, the best differential diagnostician in this case to help us out of our quandary.

During the last year I have collected and made notes of thirty cases of nervous disorders following joint affections. I have probably seen many more but have neglected to make notes of them. These notes when analyzed show that

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[1] Erb: "Handbuch der Electrotherapie," p. 201.

[2] Vierordt: "Uber atrophische Lahmungen der oberen Extrem," *D Archiv. f. klin. med.*, 1882, Bd., xxxi, p. 506.

The knee-joint was affected in	4	cases.
" wrist- " " " "	4	"
" elbow- " " " "	3	"
" finger-joints were " "	3	"
" shoulder-joint was " "	10	"
" ankle-joint " " "	2	"
" hip-joint " " "	4	"

The conclusions which I am justified in drawing from the notes of these 30 cases, are:

1st. That in all cases, except those involving the ankle- or wrist-joint, the muscles affected were the extensors of the diseased articulations.

2d. That in those cases involving the ankle or wrist, the affection was descending instead of ascending in its course, and that the extensors were not affected to any greater extent than the other muscles.

3d. That in cases of arthritis of any of the joints of the fingers, the interosse muscles suffered first and most.

The cases which merit a more detailed description and which present particular points of interest are the following:

Case 1.—Mrs. F., æt. 24. While out walking last winter she slipped, and falling, struck her right knee with considerable force. The joint rapidly increased in size and was very painful to the touch. When I examined her upon the day following the accident the knee was very much swelled, the patella pushed prominently forward, and a distinct feeling of fluctuation was obtainable. Passive and active motion was very difficult and painful. The joint affection improved rapidly; but upon the seventh day a distinct paralysis was noticeable, and whereas the movement of flexion of the leg upon the thigh was easily executed, that of extension was connected with great difficulty. The patient could only by the strongest effort of the will produce any contraction of the triceps cruris. Atrophy could, at that time, not be made out. After a lapse of two more weeks—three weeks from the time of the injury—no difference in the appearance of the affected joint itself from that of the healthy one was observable. As the effusion became absorbed, however, the paralysis increased, and it was then an impossibility for the patient, the leg having been flexed upon the thigh, to extend the same. The affected muscle remained perfectly flaccid and presented a striking contrast to the triceps of the other thigh. At this time atrophy of the muscle was well marked, showing a difference of three centimètres in favor of the healthy limb. The gluteal muscles also were somewhat involved, for upon inspection a flattening of these muscles was noticeable. The electro-contractility of the muscles was decreased to the action of both currents. There was, however, no degeneration reaction, and the contractions obtained were in their normal relationship. It required a considerable stronger current to produce contractions upon the affected muscles than upon the unaffected.

The patient was discharged cured at the end of three months. Of the treatment I shall speak later.

CASE 2.—The following case illustrates how early after the injury paralysis and atrophy may ensue. The period between the occurrence of the injury and the onset of the paralysis is, in this case, the shortest that I have ever observed.

Patient. A laborer, thirty-four years of age, was struck upon the left knee by a falling piece of timber on a Friday afternoon. He went home, and under the counsel of a physician, applied ice to the joint. The swelling, which could not have been very great, went down, and when I saw him upon the following Monday, seventy-two hours after the injury, a distinct paralysis of the triceps was noticeable, and atrophy was distinguishable upon the following Friday. Both the paralysis as well as the atrophy may have been present before the days upon which I detected them, as I saw the patient for the first time upon the day on which I noticed the paralysis, and then did not see him again until the the day upon which I made out the atrophy.

CASE 3.—The following case illustrates very well how entirely disproportionate the effect may be to the cause. It is indeed astonishing to find a periarthritis, which is almost *nil*, produce an almost entirely useless limb. In the majority of cases, it is true, the joint affection, whether it be spontaneous or traumatic in its origin, which causes the subsequent paralysis, is a painful one, and one of great severity; but there are frequent exceptions to this, and a simple sprain may produce a paralysis which is very far-reaching in its effect. This fact has induced Charcot to utter the formula: "The paralysis and atrophy following joint affections stand in no ratio whatever to the primary lesion."

Patient, L. H., æt. 30, merchant. While walking, his left ankle-joint turned so that the outer margin of the foot rested upon the ground. The pain was very severe, but he did not fall. He was, however, obliged to sit down and rest for a short time. He then continued his walk and used the joint for several hours, and then, because it felt hot and heavy, he went home and took off his shoe. Did not notice any change in the appearance of the joint, but says it was painful upon pressure. During two months his ankle seemed somewhat tender, and he would have an occasional darting pain in the foot and up the leg. Did not give up his occupation. At the end of two months he noticed a slight weakness in the injured leg. This was particularly observable on going down stairs. He then consulted various physicians, but did not place himself under any systematic course of treatment. After another lapse of two months—four months after the accident—he came under my observation. His condition was then as follows: His foot, which hangs with the toes pointing downward, cannot by any effort of the will be brought to a right-angle with the leg. When he walks—and this is a great effort for him—he does so upon his toes, and does not bring his heel to the ground. The toes may be easily raised, but they fall back again by their own weight. The interossei of the foot are atrophied, the peroneal muscles to a very great extent; and the muscles of the thigh are also involved. The gluteus maximus is evidently considerably atrophied, for a large depression takes the place of its former prominence. Added to this, severe neuralgia of the peroneal and sciatic nerves rendered his condition almost unendurable.

It is hardly necessary to cite any more cases illustrative of the various symptoms which occur during the progress of this disorder: the above three cases, together with the symptomatology, are amply sufficient; but before speaking of the pathogenesis and treatment, I desire to mention a class of cases which have been described as periarthritides. The majority of my cases in which the shoulder-joint was affected come under this category. Putnam has lately, in the *Boston Medical and Surgical Journal*, written quite an extensive article descriptive of this class. The symptoms are similar to those which I have described above, but they vary in some particulars.

There is according to Putnam, and I can corroborate it,

(1) An inability to raise the arm above the horizontal plane of the shoulder.

(2) Spontaneous pain generally worse at night, felt either in front of the joint and extending down into the biceps, or behind and remaining stationary.

(3) A slight degree of muscular atrophy of the deltoid and other muscles of the shoulder.

(4) Tenderness on pressure over certain points about the shoulder.

Duplay[1] and Desplats[2] show that pathologically this affection involves the subacromial bursa, and the loose bursoidal connective tissue beneath the deltoid muscle, and that adhesions are formed external to the joint, the surfaces of the joint itself remaining unaffected.

The pathogenesis of these nervous sequellæ of joint affections has long been a mooted question. Ever since Hunter advanced his theory, which as before remarked, explained these accidents, by a kind of sympathetic action, up to the latest theory, that of Charcot, the subject has been theorized upon and retheorized upon, until we to-day occupy almost the same position in regard to it as we did at the end of the last century. The writers who succeeded Hunter, in describing the paralysis consecutive to dislocations of the humerus, ascribed its causation to a nerve injury produced by pressure of the head of the humerus. Once that pressure was suggested as a cause, it was taken up and dilated upon ad nauseam, and pressure in its various forms became a veritable scapegoat. Roux (*Annales de Chirurgie*, 1845,) claimed that the effusion into the joint produced the pressure which caused the paralysis. After this the pressure of the apparatus applied during the treatment of the joint affection was looked upon as the producing cause. Gosselin, [1] was the first to take a decided stand against this indiscriminate pressure theory, and showed plainly by arguments which are now trite, that pressure had nothing to do with the causation of these affections. Then followed the theory of functional inertia. This starts from the principle that, the nutrition of an organ being controlled by the harmonious coöperation of all the functional and nutritive elements, if this harmony is destroyed by rest and inactivity of the affected limb, the nutrition of the muscles will necessarily suffer. This theory certainly does not accord with clinical facts, for cases are on record in which the muscles have been in absolute repose for a very long period of time, without any atrophy ensuing. Furthermore, I see no good reason, if this be true, why, in persons confined to their bed on account of some joint affection, the healthy limb is not also affected in its nutrition. It is possible that in some few cases, those for instance in which the atrophy comes on slowly and late, may be explained by this theory; but those cases, and they form by far the majority of all, where the atrophy ensues rapidly after the joint affection, can certainly not be satisfactorily thus explained.

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[1] Duplay: *Archives Gén. de Méd.*, 1872.

[2] Desplats: *Gazette Hebdomadaire*, 1878.

Vulpian was the first, in 1875, to distinctly enumerate a purely nervous theory as an explanation for these occurrences. In his "Leçons sur l' appariel vaso-moteur," Paris,

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[1] Gosselin: *Gazette Hebdomadaire*, 1859. Clinique Chirurgicale de l' hôpital de la Charité.

1875, vol. ii, in speaking of atrophy of the deltoid consecutive to arthritis of the shoulder-joint, he says: "In the majority of cases it must be classed among the atrophies called reflex; that is to say, among those which are a result of a change in such or such region of the gray matter of the spinal cord, produced by irritation of the peripheral ends of certain sensitive nerves."[1] This is the reflex theory in its entire simplicity. All writers who have written upon the subject since that time, with the exception of Charcot and Descosse, have adopted this theory and followed in Vulpian's footsteps.

Charcot, however, has so modified it that his theory is essentially a new one, and Descosse has entered upon an unbeaten path.

To these two last theories, which merit particular attention, I will now pass.

It is hardly necessary at present to show that the various component parts of a joint,—the ligaments, the synovial membrane, etc., in spite of appearing almost devoid of nerves,—have a very luxuriant supply. Sappey, in his Anatomy, speaking of the ligaments, says: "If I were to assert that they are as rich in nerves as the cutaneous covering, I would probably find myself accused of exaggeration, and, nevertheless, I would remain below the actual truth. The greater part of the ligaments is in reality richer in nerves than the skin of the trunk and of the limbs, but they have less than the skin of the fingers and toes." This supply of nerves to a joint being acknowledged, it is not difficult to admit that any inflammation which involves the joint itself may be propagated to the nerves of the joint and produce an irritation which will manifest itself by the hyperalgesia, hyperæsthesia, or neuralgia. The continuance of the cause would bring about more serious disorders, affecting the nutrition of the muscles to which the nerves are also distributed. Thus the termination of the nerve filaments distributed to a joint, participate in the general inflammation, and this is propagated from molecule to molecule, until the filaments become involved. This is the theory of Descosse, which, although not entirely satisfactory, is to my mind the simplest, and the one capable of explaining most of the symptoms.

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[1] Dans la plupart des cas, elle doit être rangee parmi les atrophies dites réflexes cést à dire parmi celles qui sout la conséquence d' une modification provoquée dans telle on telle région de la substance grise du centre nerveux cérébro-spinal, par l' irritation des extrémités peripheriques de certains nerfs sensitivs.

An autopsy corroborating this theory, by showing that a neuritis is actually produced, and then by tracing the affected nerves from the joint to the paralyzed muscles, thus clearly showing the pathological connection, would be very valuable.

The only autopsy bearing upon this matter which I have been able to find, was performed by Débove, and described in the *Progress Medical*, 1880, p. 1011, upon a subject affected with chronic articular rheumatism accompanied by great atrophy of muscle. Unfortunately, this observation is entirely valueless for our purpose, as the condition of the nerve fibres in and around the joint was entirely disregarded, and the muscular tissue alone axamined microscopically.

Charcot in a clinical lecture to his students, published in the *Progrés Medical*, 1882, p. 377, supports the following theory, and seems to consider it as the one which merits general acceptance. He says that the joint lesion, by means of the irritated articular nerves, has reacted upon the spinal centre and there modifies the centres, whence arise the motor nerves and those nerves which preside over the nutrition of the muscles.

He claims the existence in the cord of a more or less direct connection between the cells of origin of the articular nerves, and the cells of origin of the motor and trophic nerves of the extensor muscles. When other muscles besides the extensor are affected, Charcot claims that the irritation which originated in the articular nerves has spread itself by diffusion to these other muscles. His conclusion is, that in these cases we have to deal with a spinal affection, which causes the atrophy and paralysis; and that this affection consists of a kind of inertia or stupor of the cellular substance.[1]

The great variety of disorders which are encountered as sequellæ of joint affections, necessitates the command of a variety of means to combat them. The treatment naturally varies with the nature of the symptoms; and it would be utterly impossible to lay down a fixed plan of treatment as applicable to all cases. Still there are certain cardinal principles which must not be transgressed, and to those I will attract your attention. Of the treatment during the first stages of the disease, during the progress of the arthritis itself, I will say nothing, as it is not my province to discuss that, and the surgeons would probably consider it an infringement. The chief agents which we have at our disposal, in the treatment of these affections, are galvanism and faradism, massage, mechanical movements, nerve and muscle percussion, and hydrotherapy in the form of hot and cold douches. The indications for the usage of the different currents of electricity do not differ here from those which we lay down in other circumstances. There is one caution which I desire to express here, and that is, against the use of the faradic current while there are any neuralgic pains present, and I frequently interrupt the electrical treatment for a time, if these pains reappear after having been absent. In 1872, Prof. Lefort, of Paris, in a paper read before the Societé de Chirurgie, recommends very highly continuous currents of low intensity and applied for a long period of time. He claims, in cases like those under consideration, to have achieved rapid and permanently satisfactory results. He allows the current to pass uninterruptedly for days at a time; or, if the patient is not obliged to be confined to his bed, he applies the current at night only. This plan of treatment has found some adherents in France. Lefort himself admits having produced severe eschars in some instances, and for the avoidance of this, counsels interposing a wet sheet between the skin and the electrode. I have never tried this mode of treatment, and merely mention it here as an historical curiosity.

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[1] Une sorte d' inertie, de stupeur de l' élément cellulaire.

The agent, however, which in my hands has proven far more serviceable than either the galvanic or the faradic current, is massage. 'This, combined with mechanical movements, has aided me in effecting a cure in many cases in which, with electricity alone, I would have made a signal failure. Upon this question of massage, owing to its great importance in the therapeutics of this class of cases, I desire to make a few remarks. By massage I do not mean the general manipulation of the body known as hygienic massage, but local manipulation of the muscles. Massage, derived from the French verb masser,—to knead,—is really a misnomer, for the various methods of massage are numerous, and those of kneading is only one. Thus, in the treatment of the neuralgia which complicates the cases under consideration, no kneading is employed, but a rubbing or stroking; and in those cases of hypertrophy of tissue, or in those of periarticular adhesions, quite a different procedure is called into requisition. Nevertheless, this is all massage,—even the mechanical and systematic muscular exercises may be included under this name. The various effects which we can produce by massage are:

(1) The diffusion of any articular effusion.

(2) The comminution of vegetations.

(3) The loosening and destruction of adhesions.

(4) Increase of circulation.

(5) Stimulation of muscular fibres.

The fact that exudations may be diffused by means of massage has been largely disputed, and does not meet with the recognition which it deserves.

The following experiment, by v. Mosengeil,[1] proves conclusively that these denials are unfounded. V. Mosengeil made a series of experiments, of which I will give one in detail.

For these experiments he made use of a fine emulsion of the best Indian ink.

EXPERIMENT.—On Jan. 27, at 9 A. M., a thick solution of finely pulverized black Indian ink was injected by means of a hypodermic syringe into both knee-joints of a vigorous female rabbit. Not quite an entire drop of fluid returned through the puncture. The temperature in the rectum was 100.8° F. immediately after the injection. At 9:30 the right knee was massaged. At 9:45 another syringeful of finely triturated ink was injected into each knee-joint, and the right knee massaged immediately after. Thereupon the swelling went down. The left knee which, for the sake of control, was not massaged, in consequence of the movements of the rabbit also grew gradually thinner. At three o'clock in the afternoon, another syringeful was injected into each joint and the right joint again massaged. After the lapse of two minutes all enlargement had again disappeared ; the left joint did, this time, not decrease in size. The temperature had, up to that time, risen to 102.2° F., and, in the evening at 8:30, it rose to 104°. At 8:45 ink was again injected into each knee-joint, and the right one again massaged. The left joint this time did not retain an entire syringeful. On the morning of the 28th, half a syringeful of thick ink was injected into each elbow-joint of the animal, and the joints massaged immediately after. Thereupon the animal was killed and dissected. The condition was as follows : Ink was found in the periarticular connective tissue of the upper extremities, and around the punctures of the needles, permeating the subcutaneous parts, and distributed in irregular spots. The black coloring matter could be traced upward, following the course of the vessels and muscular interstices. The lymphatic glands of the axilla contained coloring matter upon the one side, and the minute afferent vessels were colored intensely black. In all the joints which had not been massaged, much more coloring matter was found than in those which had been so treated. It was thus shown that, in a few minutes after the massage, the particles of coloring matter, which had been injected into the joint, had been diffused and forced into the lymphatics.

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[1] *Langenbeck's Archiv f. klin. Chirurgie*, 1876, 9 bd., 3 und 4 heft.

One reason why the treatment by massage has met with so little support is the difficulty encountered in following the descriptions of European writers. They have divided the different methods into so many classes and varieties that it is impossible to bear them all in mind. The French writers especially have divided and subdivided, laying down a special indication for each subdivision until one is perfectly bewildered by the vast array of methods which are claimed to be essential to success.

Rossbach[1] makes a very simple division of massage into stroking, rubbing, kneading, and beating, at the same time severely criticising the inventors of the numerous subdivisions, he says: "As may be seen from the physiological part it is simply a question of the removal of blood, lymph, and exudations, from the periphery toward the centre. For this purpose therefore, stroking, rubbing, kneading, and beating, are sufficient to achieve every thing that can be achieved by this method, and therefore the urgent request might be addressed to those ingenious inventors of new diminutive subdivisions, please to keep their inventions to themselves."

The process which, in the treatment of these cases, has been of great service to me, particularly in those cases in which there is no hypertrophy of cellular tissue, is that known as muscle percussion or beating (Tapottement). For this purpose I have been in the habit of using the following two instruments. It is well to mention here that this is the only form of massage which I consider can be as well performed by means of mechanical contrivances as by the hand; for all other modes the hand certainly merits the preference. The first was invented by C. Klemm, manager of a gymnasium in Riga, and consists simply of elastic rubber hollow rods attached to a handle. They are manufactured in three different sizes, ranging in length and thickness. The second, and I believe I am indebted to Dr. Hammond for this idea, is nothing more nor less than a baby's rattle, filled with cotton, and used as a hammer. Both of these instruments are only serviceable when the tissues to be acted upon are very superficial.

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[1] "Lehrbuch der Physikalischen Heilmethoden," p. 492.

The majority of writers, during the early stages of these disorders, imperatively counsel rest. Once that the active inflammation has subsided, I am entirely opposed to this. I generally begin a course of passive motion at once, break up all periarthritic adhesions mechanically, and if there is paralysis or atrophy, without any neuralgic pains, I make use of faradism combined with some form of massage. I consider mechanical movement passive,—and, if possible, also active,—a very important adjutvant to the application of massage, for Ludwig has shown that the action of the muscles upon the fluids of the body is comparable to that of a pump. Therefore, admitting that the extraneous pathological product, which has been formed in and around the joints in consequence of the inflammation, has been loosened and triturated by means of the massage, and being then in a condition suitable for removal by the lymphatics, it is then compressed by means of the muscular action, which is produced either actively or passively, and forced into the efferent vessels. If, however, there is pain along the course of the nerves, I discontinue the electricity, and having satisfied myself that the pains are neuralgic, and not due to a neuritis, I proceed with the massage alone. As far as the treatment of the paralysis and atrophy is concerned, I would be at a loss to state in which I have most confidence —electricity or massage; but it is certain that in many cases neither one nor the other will suffice, but that the two combined, together with mechanotherapy and hydrotherapy, are necessary to bring about satisfactory results. All these agents seem here to act similarly, by stimulating the nutrition of the affected muscles and surrounding parts, by increasing the flow of blood, and perhaps thus causing a reflex excitability of the motor tracts.

The prognosis of these cases under treatment is good, still, occasionally cases are encountered where the utmost patience and perseverence are essential to success, and for this class of cases I can give no better advice than is contained in the proverb, "Gutta Cavet Lapidem."